2. Collins R, Keech A, Peto R, Sleight P, Kjekshus J, Wilhelmsen L, et al. Cholesterol and total mortality: need for larger trials. BMJ 1992;304: 1689.
3. http://www.ctsu.ox.ac.uk/pressreleases/2001-11-13/life-saver-world-slargest-cholesterol-lowering-trial-reveals-massive-benefits-for-high-riskpatients/?searchterm=merck (accessed Oct 19, 2007).
4. Boden WE, O'Rourke RA, Teo KK, Hartigan PM, Maron DJ, Kostuk WJ, et al.; COURAGE Trial Research Group. Optimal medical therapy with or without PCI for stable coronary disease. N Engl J Med 2007; 356:1503-16.
5. Strandberg TE, Pyörälä K, Cook TJ, Wilhelmsen L, Faergeman O, Thorgeirsson G, et al.; 4S Group. Mortality and incidence of cancer during 10-year follow-up of the Scandinavian Simvastatin Survival Study (4S). Lancet 2004;364:771-7.
6. The Long-Term Intervention with Pravastatin in Ischemic Disease (LIPID). Study Group. Prevention of cardiovascular events and death with pravastatin in patients with coronary heart disease and a broad range of initial cholesterol levels. N Engl J Med 1998;339:1349-57.

doi:10.1016/j.jvs.2007.10.065

Reply

Previously published results from the Heart Protection Study (HPS) show clearly that lowering LDL cholesterol by about 1 mmol/L (38 g/dL) with simvastatin produced a highly significant 13% (SE 4) relative reduction in all-cause mortality (1328 [12.9%] simvastatin-allocated vs 1507 [14.7%] placebo-allocated deaths; $P = .0003$) during the scheduled 5-year treatment period.[1] This very definite survival benefit reflected the combined impact of a highly significant 17% (SE 4) relative reduction in vascular deaths (781 [7.6%] vs 937 [9.1%]; $P < .00001$) and of a nonsignificant difference in nonvascular mortality (547 [5.3%] vs 570 [5.6%]; $P = .4$). A subsequent report showed that there were similar relative reductions in vascular deaths (and nonfatal major vascular events), with no evidence of any adverse effects on nonvascular deaths (or cancers), in a range of different circumstances (including among women).[2] The reduction in vascular mortality started to emerge during the first year of statin treatment and increased during each subsequent year of treatment, with no adverse effect on nonvascular mortality emerging during or after the scheduled treatment period.[1-3]

Meta-analyses of individual patient data from large randomized trials (including HPS) have reliably demonstrated that statin therapy reduces vascular mortality substantially, while producing little or no effect on nonvascular mortality.[4] Consequently, the relative reduction in all-cause mortality in some particular circumstance is determined not only by the size of the relative reduction in vascular mortality with statin therapy but also by the ratio of vascular to nonvascular deaths. Moreover, separate assessment of the effects of statin therapy on vascular mortality and on nonvascular mortality in such circumstances (considered in the context of the overall findings for cause-specific mortality and for the much larger numbers of nonfatal vascular and nonvascular events) is likely to provide a more sensitive assessment of any benefits and hazards than would direct comparisons of deaths from all-causes.

With regard to the subgroup of patients in HPS with peripheral arterial disease (PAD), the observed 10% (95% CI -5-12) relative reduction in vascular mortality (10.2% simvastatin vs 11.2% placebo) was not significantly different from the 23% (12-32) relative reduction observed among the other high-risk patients studied (heterogeneity P value = .1). Moreover, this lack of heterogeneity of benefit with statin therapy was reinforced by the similar (heterogeneity P value = .5), and highly significant, relative reductions in major vascular events (MVE): (ie, vascular deaths, heart attacks, strokes, and revascularizations) among patients with PAD (22% [SE 4]; $P < .0001$) and the other high-risk patients (25% [SE 3]; $P < .0001$).[5] The apparent lack of effect on the small number of aneurysm deaths or repairs should be considered in the context of these large reductions in vascular events. In terms of the absolute benefits in HPS, allocation to statin therapy prevented 63 (11) first MVEs, and 116 (21) first and subsequent MVEs, per 1000 PAD patients. This corresponds to a "number needed to treat" to prevent a first MVE of 16 (SE 3), although this underestimates the benefit of actually taking a statin because only about two-thirds of patients complied with their allocated treatment during the 5-year study period. In terms of safety, as was observed overall in HPS, there was no apparent effect on nonvascular mortality among the patients with PAD (7.3% simvastatin vs 7.7% placebo; hazard ratio 0.94; 95% CI 0.79-1.12; P = NS).

In conclusion, HPS has shown that 40 mg simvastatin daily safely reduces both vascular mortality and major vascular morbidity in patients with PAD and in other high-risk patients, without adverse effects on nonvascular mortality or major nonvascular morbidity. It is, therefore, entirely reasonable to conclude that vascular surgeons should consider statin therapy for all patients with proven PAD irrespective of age, gender, and baseline lipid levels.

Richard Bulbulia
Louise Bowman
Karl Wallendszus
Sarah Parish
Jane Armitage
Rory Collins

CTSU, University of Oxford
Oxford, United Kingdom

REFERENCES

1. Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20,536 high-risk individuals: a randomized placebo-controlled trial. Lancet 2002;360:7-22.
2. Heart Protection Study Collaborative Group. The effects of cholesterol lowering with simvastatin on cause-specific mortality and on cancer incidence in 20,536 high-risk people: a randomized placebo-controlled trial. BMC Med 2005;3:6.
3. Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study (HPS): mortality, cancer incidence and major vascular events during 4 years post-trial follow-up of HPS. JACC 2007;49:393A.
4. Cholesterol Treatment Trialists' (CTT) Collaborators. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90,056 participants in 14 randomized trials of statins. Lancet 2005;366:1267-78.
5. Heart Protection Study Collaborative Group. Randomized trial of the effects of cholesterol-lowering with simvastatin on peripheral vascular and other major vascular outcomes in 20,536 people with peripheral arterial disease and other high-risk conditions. J Vasc Surg 2007;45:645-54.

doi:10.1016/j.jvs.2007.11.061

Regarding "Light assisted stab phlebectomy: Report of a technique for removal of lower extremity varicose veins"

In the article by Lawrence and Vardanian,[1] the authors reported on light assisted stab phlebectomy. I was amazed by how a simple procedure can be made so eloquently complicated. We have performed stab avulsion phlebectomies on more than 2000 patients in our office without sedation. We use 1% local lidocaine anesthetic injected only into the site of the 2 mm puncture over the previously marked vein. Tumescent anesthesia is not required or used since we find that it oozes from the incision making application of Steri-strip closure unreliable. Provided that the vein itself is removed, without any adjacent subcutaneous tissue, patients feel no pain. At most, they notice a pulling sensation. Simple finger pressure for a minute or two prevents bleeding even from a large varicosity.

No sutures are required to close the incision but simply Steri-strips. We do not wrap the leg but rather use a 30 to 40 mm